

Cerebral microembolization after protected carotid artery stenting in surgical high-risk patients: Results of a 2-year prospective study

Frank D. Hammer, MD,^a Valerie Lacroix, MD,^b Thierry Duprez, MD,^a Cecile Grandin, PhD,^a Robert Verhelst, MD,^b Andre Peeters, MD,^c and Guy Cosnard, PhD,^a *Brussels, Belgium*

Background: This was a prospective single-center study to assess and analyze cerebral embolization resulting from carotid artery stenting with neuroprotective filter devices in patients considered as poor surgical candidates for surgical carotid endarterectomy.

Methods: Fifty-three consecutive patients with an internal carotid artery stenosis were treated by placement of carotid Wallstents with two different types of temporary distal filter protection devices: the Spider filter and the FilterWire. Diffusion-weighted magnetic resonance imaging (DWI) of the brain was obtained 24 hours before the procedure and within 5 to 30 hours after the procedure to detect ischemic brain lesions resulting from the procedure. Inclusion criteria were symptomatic ($\geq 70\%$) or asymptomatic ($\geq 80\%$) stenoses in surgical high-risk patients.

Results: Two (4%) regressive minor strokes occurred. Postprocedural DWI detected new focal ischemic lesions in 21 patients (40%). The average number of lesions was 5.9 per patient, and the mean lesion volume was 1 mL or less in 19 patients (90%). Small differences were found in the lesion distribution: homolateral anterior circulation in eight cases (15.1%), other vascular territories in seven cases (13.2%), and homolateral anterior circulation plus other vascular territories in six cases (11.3%). The microembolization risk seemed unpredictable on the basis of clinical parameters and internal carotid artery lesion characteristics. An increased risk in the rate of ipsilateral hemispheric embolization has been observed in difficult carotid arch implantations ($P = .04$).

Conclusions: The incidence of new focal ischemic lesions detected by DWI is higher than expected on the basis of previous reports. Embolization from the aortic arch or common carotid arteries could account for most of those events in patients considered as surgical high-risk patients. Although 90% of the events were clinically silent, this high rate of microembolization raises questions about the possible consequences on cerebral cognitive functions. (*J Vasc Surg* 2005;42:847-53.)

Stent implantation for the treatment of extracranial carotid artery stenosis has become an alternative to carotid endarterectomy (CEA). During those procedures, embolization of air, thrombus, or plaque material can be responsible for neurologic complications. Protection devices placed distally to the lesion, before stent deployment and balloon dilation, have been developed to minimize this potential risk. The exact risk of embolization still remains controversial and depends on multiple factors. The purpose of this prospective study was to determine the incidence of new areas of cerebral ischemia by diffusion-weighted magnetic resonance imaging (DWI) in high-surgical-risk patients resulting from carotid artery stenting (CAS) and to analyze the lesion distribution and the clinical and procedural factors that could explain those events.

METHODS

Patient selection. This prospective study was started in October 2002. The protocol was proposed to patients with major cardiovascular risk factors and symptomatic

internal carotid artery (ICA) stenosis of 70% or more or asymptomatic stenosis of 80% or more. One or more of the following inclusion criteria had to be present: two or more coronary vessels with 70% stenosis or more, ejection fraction less than 30%, a New York Heart Association functional class of III or higher, bronchopulmonary obstructive disease, restenosis after a previous CEA, previous radical neck surgery or radiation therapy, surgically inaccessible lesions, and contralateral occlusion of the ICA.

Exclusion criteria were an evolving stroke or a recent stroke within the previous 7 days, a transient ischemic cerebral event within the last 24 hours, a history of allergic reaction to any study medications or material, renal insufficiency (creatininemia > 2 mg/dL), bilateral occlusions of the iliac arteries or abdominal aorta, totally calcified ICA stenoses, or suspicion of fresh thrombus at the level of the ICA stenosis. Informed consent was obtained from each patient.

Carotid artery imaging. All carotid lesions were detected and imaged by color Doppler ultrasonography (CDUS) with a ATL 5000 (Philips Medical Eindhoven, The Netherlands) by using high-frequency probes. The degree of stenosis was quantified on the basis of the peak systolic velocity measured at the level of or immediately after the stenosis (velocity > 210 m/s, corresponding to $\geq 70\%$ stenosis). Preprocedural diagnostic imaging of the arch vessels and carotid stenosis by magnetic resonance (MR) angiography was optional.

From the ^aDepartment of Radiology, ^bCardio-Vascular Surgery, and ^cNeurology, University Hospital St Luc.

Competition of interest: none.

Reprint requests: Frank D. Hammer, MD, Department of Radiology, University Hospital St Luc, 10 Av Hippocrate, Brussels 1200, Belgium (e-mail: hammer@rdgn.ucl.ac.be).

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Digital subtraction angiography (DSA) of the carotid bifurcation, the intracranial portion of the ICA, and the brain circulation was performed in all cases (Philips Integris 3000; matrix 1024×1024). Final quantification of the ICA stenosis was performed according to the North American Symptomatic Carotid Endarterectomy Trial guidelines.¹ The visualization or absence of visualization of the anterior and posterior communicating arteries was described, and flow direction was determined before and after stenting. Contralateral common carotid artery (CCA) injections were performed in patients requiring quantification of bilateral lesions. In most patients, a global aortic arch injection was performed ($\pm 30^\circ$ left anterior oblique view) before selective carotid catheterization. A retrospective analysis of those DSA images allowed detection of severe CCA angulations and quantification of the angle of the CCA emergence with respect to the aortic arch. This angle was measured at the intersection between a centerline drawn through the first centimeters of the target CCA and a line drawn between the anterior wall of the innominate trunk and the posterior wall of the left subclavian artery, at their ostial level. An angle less than 45° was considered to be less favorable and technically more difficult for selective catheterization and sheath placement.

Comparative review of the DSA and CDUS images allowed description and classification of the carotid lesions according to the lesion length (≤ 10 or > 10 mm), presence of ulcerations (0, nonulcerated; 1, 1 mm of ulceration; 2, more than one ulceration or ulceration ≥ 2 mm), and degree of calcification (0, homogenous and noncalcified; 1, minimal calcification or $< 30\%$ calcification; 2, more than 30% calcification).

MR imaging. All MR examinations were performed on a 1.5-T MR system (Intera; Philips Medical) by using a standardized protocol including a fast spin echo/fluid-attenuated inversion recovery sequence (repetition time, echo time, and inversion time of 1000, 120, and 2100 milliseconds, respectively; field of view, 230 mm) and an echo planar imaging/spin-echo diffusion-weighted sequence (repetition time and echo time of 5110 and 84 milliseconds, respectively; field of view, 220 mm; matrix, 95×256) with and without application of diffusion-sensitizing gradients in the three orthogonal directions, performed at $b = 1000 \text{ s/mm}^2$. A set of 24 axial transverse images (5-mm thickness; 1-mm interslice gap) in similar slice locations were obtained for both sequences. A baseline MR examination was performed the day before the procedure, and a follow-up MR study was performed on the same day or the day after stent placement. Three trained neuroradiologists, blinded to clinical status and findings, independently rated the diffusion-weighted trace images for the absence or presence of acute ischemic parenchymal damage. If positive, lesions were quantified by using the following scoring system: number of lesions, location of lesions, lesion sizes (categorized into lesions < 5 mm, 5–10 mm, or > 10 mm), and total lesion volume (milliliters). Locations were described to determine the vascular territories (anterior or posterior circulation), the side (ipsilateral or con-

tralateral to the ICA stenosis), and the distribution (cortical, subcortical, or deep areas). In addition, the baseline preprocedural fluid-attenuated inversion recovery images were reviewed to assess pre-existing chronic or subacute infarcts and the degree of cerebral leukoaraiosis, which has been evaluated according to a 0- to 9-point scale described by Manolio and colleagues² and Longstreth and associates,³ by using a template image and a text description.

Endovascular procedure and clinical assessment. CAS was performed by two experienced interventionalists (F.D.H. and R.V.), assisting each other. Two types of filters were used: the FilterWire (EX and EZ) from Boston Scientific (Maastricht, The Netherlands), composed of a perforated polyurethane membrane attached to a nitinol loop, and the Spider filter (ev3 Inc, Paris, France), comprising a nitinol basket. The outer diameters of the filter delivery catheters were 3.9F, 3.5F, and 2.9F for the EX, EZ, and Spider filters, respectively. The filter pores were less than $110 \mu\text{m}$. The Spider filter was mainly selected for large ICA diameters because of the availability of larger devices (5–7 mm). After filter placement only, the ICA stenoses were dilated (percutaneous transluminal angioplasty) before stent deployment by using 3- or 3.5-mm-diameter monorail balloons (Gazelle or Ultra-soft SV; Boston Scientific) in cases judged to be too tight or tortuous, to allow primary passage of the stent. Monorail 5F self-expandable carotid Wallstents (Boston Scientific) were placed in all patients and were chosen according to the lesion's length and the ICA and CCA diameters, usually covering the external carotid artery. After deployment, stents were always postdilated (4–6 mm). The balloon was chosen according to the CDUS measurements; the ICA diameter was never oversized beyond the stenosis.

Gross eye inspection of the filter determined the number and size of debris captured (0.5–1 mm or > 1 mm). Patients were premedicated with clopidogrel 75 mg/d (Plavix, Sanofi-Synthelabo, Belgium), acetylsalicylic acid 160 mg/d (Asaflo, Sandipro, Belgium) for at least 3 days before the procedure. Clopidogrel (75 mg/d) was prescribed for 2 months and was discontinued only in patients scheduled for an operation, 10 days before the intervention. Acetylsalicylic acid was not discontinued and was prescribed for a lifetime. During the procedure, a bolus of 5000 U of heparin was administered, and additional heparin (up to 5000 U) was injected, depending of the activated clotting time (250–300 seconds). Per-procedure patient monitoring and management was ensured by an anesthesiologist. Puncture site closure devices were used in all cases (Perclose and Angioseal), thus allowing uninterrupted antiplatelet therapy.

An independent neurologist performed neurologic examination the day before the intervention and within 24 hours after the intervention. Clinical re-evaluation of the patient and control CDUS were scheduled at 1 and 6 months and then annually.

Statistical analysis. Values are expressed as mean \pm SD. For analysis of descriptive statistics and continuous and categorical data, Pearson χ^2 or Fisher exact tests were used as appropriate. A P value $< .05$ was considered significant.

All statistical analyses were performed with SPSS version 11.5 software (SPSS Inc, Chicago, Ill).

RESULTS

Between November 2002 and January 2005, 68 patients fulfilled the preliminary clinical and CDUS selection criteria and were scheduled for angiography and possible stenting. During DSA, 5 patients were excluded: CDUS overestimation of the stenosis ($n = 2$), severe tortuosity of the CCA ($n = 1$), and complete calcification of the ICA lesions ($n = 2$). In another patient the procedure was attempted, but the protective filter could not be placed because of an ICA kink. Protected CAS was successfully performed in 62 patients (98.4%). DWI studies were obtained in 53 patients. The reasons for missing MR imaging (MRI) data were incomplete MRI studies ($n = 3$) and pacemakers contraindicating MRI ($n = 6$).

Patient data and technical considerations. The mean age of the 53 patients with DWI studies was 72.3 ± 9.2 years (range, 50-92 years). The male-female sex ratio was 3:1. Atherosclerotic risk factors included a history of tobacco smoking (59%), hypertension (89%), diabetes type 1 (2%), diabetes type 2 (38%), and treated dyslipidemia (74%). Mild chronic renal insufficiency, defined as creatinemia of 1.5 to 2 mg/dL, was found in six patients (11%).

Eighteen (34%) symptomatic carotid stenoses were treated. In addition to systemic risk factors, the following unfavorable conditions for surgery could be retained in these patients: 50% to 70% contralateral ICA stenosis ($n = 6$), contralateral ICA occlusion ($n = 2$), hostile neck ($n = 1$), and coronary artery bypass planned within 4 weeks ($n = 2$).

The remaining 35 patients (66%) were asymptomatic but presented tight ICA stenoses ($\geq 80\%$) and the following clinical situation: hostile necks (radical neck surgery and radiotherapy, $n = 7$), restenosis after CEA ($n = 2$), surgical failure of CEA (high cervical carotid bifurcation; $n = 1$) in 29%, and a major operation planned (coronary bypass, $n = 14$; aortic valve replacement, $n = 1$; abdominal aortofemoral bypass, $n = 1$) in 46% of the cases. Twelve asymptomatic patients (34%) had a contralateral ICA stenosis of more than 50%, and three (9%) had a contralateral ICA occlusion.

The mean ICA stenosis rate was $87\% \pm 6\%$, and 58% of the patients had stenosis graded as $\geq 90\%$. Carotid Wall-stents were easily deployed in all cases except in one patient, in whom the stent-covering membrane ruptured during initiation of the deployment and necessitated retrieval of the partially uncovered stent and placement of a new stent. In one patient, two stents had to be used because of a severe stenosis of the mid portion of the CCA associated with the ICA stenosis. All patients had good improvement of the ICA lumen, although minimal residual stenosis of 15% to 25% could be observed in six cases and 35% stenosis was observed in one case.

The following protective filtering devices were used: FilterWire ($n = 41$) or Spider filter ($n = 12$). Debris in the protective filters (0.5-1.5 mm) was found in 36 cases (68%). Small fragments (<1 mm) were found predominantly

(72%), and the average number of fragments was fewer than 3 in 44% of the cases, 3 to 6 in 25%, and more than 6 in 31%. The mean duration of the procedure, from the local anesthesia to access closure, including diagnostic angiography, was 48 ± 12 minutes.

Clinical results. No major systemic complications occurred during the procedures or the first 30 days of follow-up. One patient developed a puncture site hematoma, which resolved spontaneously. No cardiac infarcts or deaths were observed.

Neurologic events within 30 days were detected in two cases (4%). Both patients belonged to the symptomatic group, and the deficits were categorized as minor reversible strokes.

One patient presented with a quadrantanopsia, detected 2 days after CAS. Three large lesions were found with DWI in the left contralateral occipital lobe (ischemic volume, ± 4.7 mL). Symptoms completely regressed within 10 days, and the visual field was normal 1 month after the procedure. The second patient presented signs of confusion and contralateral hemiparesis immediately after stent placement and dilation. DWI detected multiples small lesions ($n = 25$; total volume, 2 mL) exclusively located in the ipsilateral cerebral hemisphere; the largest measured 13 mm in diameter. The arm weakness resolved almost completely within 3 weeks (nondisabling stroke). The functional health index, by using the modified Rankin scale,⁴ was categorized by the neurologist initially at 2 and one month later at 1. At 1 year, the patient had completely recovered.

Patients were followed up by clinical and CDUS examinations (1-24 months; median, 9 months). No patient died or developed neurologic symptoms. Intrastent hyperplasia was found in 3 (5.7%) of 53 patients: 1 patient with a 50% stenosis, 1 with a 50% to 60% stenosis left untreated, and 1 with an 80% stenosis that required additional balloon dilation and stenting.

Results of DWI. Analysis of the baseline MRI studies revealed white matter abnormalities in 50 (94%) of 53 patients. Leukoariosis was graded as mild (1-3) in 35 patients (70%) and as greater than 3 in 15 patients (30%). Sequelae of previous infarcts were found in 15 patients (28%). Focal subacute or acute DWI abnormalities were detected before the CAS procedure in 10 patients (19%). These findings confirm that a baseline study before the procedure is mandatory.

Comparison between the DWI images acquired before and after the stent procedure (mean delay, 19.3 hours) identified new ischemic brain lesions in 21 patients (39.6%). Cerebral distribution of the lesions (Fig 1) according to the side of the treated ICA stenosis into the homolateral anterior circulation (HC), contralateral anterior circulation (CC), and posterior circulation (PC) was as follows: HC, $n = 8$; HC plus CC, $n = 4$; HC plus CC plus PC, $n = 1$; HC plus PC, $n = 1$; CC, $n = 3$; PC, $n = 3$; and CC plus PC, $n = 1$. This means that eight patients (15%) had ipsilateral lesions, six patients (11%) had lesions in the ipsilateral brain and other vascular territories, and seven patients (13%) had lesions in vascular territories that were not directly linked to the treated vessel. These events could result from endovas-

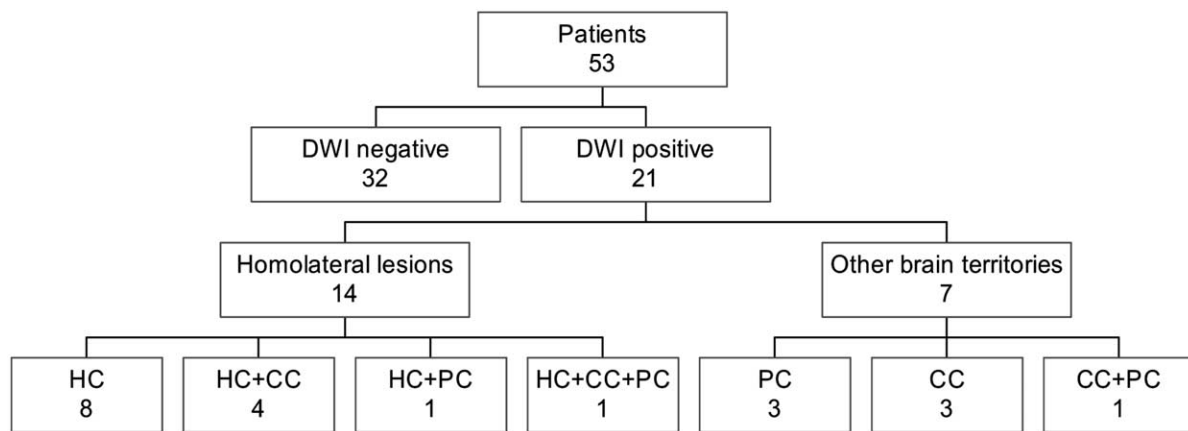


Fig 1. Distribution of the new focal cerebral ischemic lesions detected by diffusion-weighted magnetic resonance imaging (DWI) into the homolateral (HC), contralateral (CC), and posterior (PC) circulation, according to the side of the treated internal carotid artery.

cular maneuvers in the aortic arch or the contralateral CCA, from embolus migration through collateral pathways (occipitovertebral and ophthalmic anastomosis of the external carotid artery), or through the circle of Willis.

On the angiograms, a functional anterior communicating artery with an ipsilateral to contralateral flow was visible in nine cases. In only one of those patients, who also had a contralateral ICA occlusion, DWI lesions were found in the frontal contralateral brain; this definitively allowing us to conclude an embolus migration through the anterior communicating artery. Anterior to posterior flow in the posterior communicating artery, arising from the ipsilateral ICA, was visible in 16 cases. In only one of them was a single PC DWI lesion found. From these data, we can conclude that most microembolization in the part of the brain other than the ipsilateral hemisphere results predominantly, if not exclusively, from endovascular maneuvers in the aortic arch or from catheterization of other arch vessels.

The overall average number of lesions per patient with positive DWI examinations was 5.9 (HC, 5.3; CC, 3.1; and PC, 3.5). If 2 patients with massive microembolization (25 and 56 lesions) were excluded, this rate decreased to 2.2 (range, 1-6). The distribution of focal lesions into HC, CC, and PC was 60%, 23%, and 17% and 46%, 32%, and 22%, respectively, depending on whether the two patients were included or excluded. Lesions were located in cortical territories (62%), subcortical territories (34%), or deep regions (4%). The diameter of the lesions was 10 mm or smaller, and the total lesion volume was 1 mL or less (area \pm 100 mm²) in 19 (90%) of 21 patients (the 2 exceptions were the patients who had minor strokes).

Statistical analysis. Although this finding was statistically nonsignificant, we could observe a slightly higher rate of brain lesions in symptomatic patients, older patients, patients with pre-existing leukoariosis, longer procedural times, procedures performed with the FilterWire or with balloon predilation, and stenoses with low calcification (Tables I and II). We found a trend for contralateral

embolization in patients who underwent catheterization of the opposite CCA ($n = 18$) for diagnostic purposes: 33% vs 9.4%, respectively ($P = .06$).

The retrospective angiographic analysis of the CCA implantations and tortuosities, which was possible for a subgroup of 37 patients (similar positive DWI rate of 40.5%) who had had a global arch injection before selective catheterization, seems to corroborate those findings. Fourteen CCAs ipsilateral to the treated ICAs were categorized as technically more challenging for selective catheterization and subsequent sheath placement: 12 patients with a CCA-arch angle of 45° or less plus 2 patients with severe angulations of the proximal portion of the CCA (angle of 90° and 103°). The risk of ipsilateral hemispheric lesions (HC only) in those 14 patients was significantly higher compared with that in the other 23 patients: 36% vs 5% ($P = .04$; Fisher exact test).

Surprisingly, the incidence of ipsilateral brain lesions was almost identical in the patients with empty filters and in those in whom debris was retrieved (24% vs 28%). This reinforces the impression that filters are effective in preventing embolic migration in most cases and that most embolic events probably result from maneuvers in the aortic arch or CCAs, before the filter is opened, and before the CAS procedure itself.

DISCUSSION

DWI is the most powerful tool for the detection of focal brain ischemia in the acute stage.⁵ It has been used for the detection of cerebral embolism after acute ischemic neurologic events and for the detection of silent ischemic brain lesions after diagnostic cerebral angiography,^{6,7} coronary bypass surgery,⁸ CEA,⁹⁻¹³ and stenting.¹⁴⁻¹⁸ The hyperintense DWI lesions vanish over 2 weeks and may reappear as hypointense sequelae lesions thereafter.¹⁹ Optimal timing of the DWI is therefore critical.

DWI of the brain has already been used in various circumstances after surgical or endovascular procedures.

Table I. Analysis of potentially predictive factors for an increased risk of cerebral embolization during angiography and the carotid artery stenting procedure

Variable	No. Patients	DWI positive	DWI negative	% Positive DWI	P value
Symptomatic patients	18	9	9	50%	NS
Asymptomatic	35	12	23	34%	
Age <75 y	32	10	22	31%	.1
Age ≥75 y	21	11	10	52%	
Cerebral leukoariosis grade <3	31	11	20	35%	NS
Cerebral leukoariosis grade ≥3	22	10	12	45%	
Diabetic	22	8	14	36%	NS
Nondiabetic	31	13	18	42%	
Procedure time <50 min	30	10	20	33%	NS
Procedure time ≥50 min	23	11	12	48%	
Single CCA injection	13	4	9	31%	NS
Arch and/or contralateral carotid DSA	40	17	23	43%	
Contralateral stenosis (50%-100%)	23	9	14	39%	NS
No contralateral stenosis	30	12	18	40%	
Angiographic acquisitions <11	27	10	17	37%	NS
Angiographic acquisitions >11	26	11	15	42%	

DWI, Diffusion-weighted magnetic resonance imaging; NS, not significant; CCA, common carotid artery; DSA, digital subtraction angiography.

DWI was considered positive if at least 1 new lesion was found in the brain (total, 21 of 53 cases), independently from the localization (anterior or posterior circulation). Statistical analysis was based on χ^2 and Fisher exact tests (nonsignificant, $P > .05$).

Table II. Analysis of factors that are theoretically able to increase the risk for embolization from the internal carotid artery stenosis

Variable	No. Patients	DWI (+) ipsilateral	DWI (−) ipsilateral	% Positive DWI
Symptomatic patients	18	6	12	33%
Asymptomatic patients	35	8	27	23%
FilterWire	41	12	39	29%
Spider filter	12	2	10	17%
PTA before stent placement	18	6	12	33%
No PTA before stent placement	35	8	27	23%
Stenosis 70%-89%	22	5	17	23%
Severe stenosis ≥90%	31	9	22	29%
Stenosis length ≤10 mm	21	6	15	29%
Stenosis length >10 mm	32	8	24	25%
Nonulcerated stenosis	20	5	15	25%
Ulcerated stenosis grade 1-2	33	9	24	27%
Low plaque calcification (<30%)	32	10	22	31%
High plaque calcification (≥30%)	21	4	17	19%
Debris in the basket	36	10	26	28%
No debris in the filter	17	4	13	24%

DWI, Diffusion-weighted magnetic resonance imaging; PTA, percutaneous transluminal angioplasty.

Only ipsilateral brain lesions were recorded (total, 14/53 cases).

Cerebral diagnostic angiography by itself is associated with an incidence of an estimated 9% of asymptomatic cerebral infarctions.⁶ In the endovascular field, DWI has been initially used after unprotected CAS. Van Heeswijk et al¹⁵ found lesions in 15% and only in the ipsilateral hemisphere. Jaeger et al¹⁶ published a rate of 29% of new ipsilateral lesions and 9% of contralateral lesions in 20 (29.9%) of 67 patients, of whom only 1 had a neurologic deficit. The same group published the first results with protected CAS.¹⁷ They found an incidence of 15%, but in this series of 20 patients only, high-grade ICA stenosis of more than 90% was considered an exclusion criterion for the use of a filter device. Finally, Schluter et al¹⁸ found new ischemic foci in 23% of his 42 patients treated by CAS with 6 different types

of cerebral protection devices. To our knowledge, there is only 1 limited but recent study of 21 nonconsecutive patients; it reports a higher rate of 43%, similar to our results.²⁰

Team experience, endovascular material, procedural steps, medication (anticoagulation and antiaggregation), and MRI parameters have been rigorous and standardized as much as possible in our study, in accordance with other protocols or guidelines, and are not able to explain those high rates. Supporting this is our low neurologic complication rate of 4%, which seems comparable to results of CAS studies using protective filter devices in high-surgical-risk patients.^{21,22} In addition, we have recently applied an identical MRI and interpretation protocol (nonpublished data) in a limited number of surgi-

cal patients (18 low-risk patients, 78% of whom were symptomatic; mean age, 70 years) treated by CEA and found a low rate of silent embolizations (6%). This is in accordance with most surgical studies,⁹⁻¹² which have found new lesions, located exclusively in the ipsilateral hemisphere, in less than 12% of the cases and which tend to demonstrate that our imaging and interpretation protocol was not too sensitive.

In conclusion, our study, which revealed new ischemic lesions in 40% of patients, suggests that the microembolic risk could have been underestimated, at least in some subgroups of patients. Of interest is that in 13 (24.5%) of the 53 patients, or 13 (62%) of the 21 positive DWI cases, embolic lesions were found outside the vascular territory of the treated ICA. Lesions in the contralateral hemisphere could not be explained by collateral arterial pathways except in one or two patients. On the basis of our data, the main determinant for such microembolic events could be the patient's underlying diffuse atheromatosis or embolization resulting mainly from the catheter, wire, or sheath manipulation in the aortic arch and the CCAs. Great care should therefore be taken, especially during the initial phase of the procedure, before the filter placement and CAS procedure itself. Contralateral CCA angiography should be restricted as much as possible. Considering this, a direct puncture technique of the CCA (excluding hostile necks) to gain access to the targeted ICA could be a valuable technique, at least for difficult carotid accesses, but this would first require the development of an adequate puncture site closure device.

Although the vast majority of those embolic events (90%) did not induce evident neurologic deficits, the significance of such clinically silent embolization during CAS has not yet been established and should be a concern in an elderly population, especially in patients with impaired brain function or previous infarcts. A recent large population-based study²⁵ has shown that silent brain infarcts seen on DWI are associated with a steeper decline in cognitive function and may be associated with dementia.²³

Neuropsychometric deteriorations have definitively been described after CEA²⁴⁻²⁷ and cardiac surgery.²⁸ Those deteriorations, which may be definite or last for several months, could be induced by microembolization, by transient brain ischemia, or by postprocedural hyperperfusion. Data for CAS are lacking, and it remains therefore not proven which technique carries the highest risk for cognitive function alteration. Therefore, we believe that besides other short- or long-term issues, the potential consequences of microembolization resulting from CAS still need to be clarified by controlled and randomized studies before CAS can be recommended as the primary treatment option for carotid stenoses.

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INVITED COMMENTARY

Mark C. Myers, Richard J. Powell, MD, Lebanon, NH

Using diffusion-weighted magnetic resonance imaging (DWI), Hammer et al have demonstrated an alarming rate (40%) of new microinfarcts after filter-protected carotid artery stenting (CAS). These data support previous studies that have demonstrated new microinfarcts in 20% to 43% of patients after CAS.^{1,2} They are to be commended on their overall results: there were only two (4%) minor strokes that resolved completely, no myocardial infarctions, and no deaths. The pattern of these cerebral infarcts suggests that they are not due to failure of the embolic protection device. Ipsilateral DWI infarcts occurred with equal frequency in cases with and without visible debris captured in the filter. More importantly, the DWI lesions frequently involved the contralateral cerebral hemisphere, thus suggesting that many occurred before cannulation of the target carotid artery. The occurrence of these DWI lesions seems, on the basis of Hammer and associates' analysis, to correlate with difficult arch anatomy, supra-aortic carotid tortuosity, and the performance of contralateral brachiocephalic selective catheterization. Thankfully, most of these lesions seem clinically silent, but this would be better evaluated with formal neuropsychiatric testing. These results argue against routine contralateral diagnostic arteriography as part of a CAS procedure, especially because one of the two strokes did occur in the contralateral occipital lobe. In our center, like many, we are careful to minimize manipulations in the aortic arch and attempt to cannulate only the

target carotid artery during CAS. Proper patient selection and technical skill will be critical in decreasing CAS neurologic event rates to those seen with carotid endarterectomy, especially in older patient populations, in which CAS stroke and death rates have exceeded 12%.³ We use computed tomographic angiography to screen patients for difficult arch anatomy, evaluate aortic arch plaque burden, and evaluate the carotid lesion length and calcification. We have used this information to exclude some patients from further consideration for CAS on the basis of anatomic concerns. This study by Hammer et al supports the concepts that minimal, focused catheter manipulation within the aortic arch and careful patient selection based on evaluation of aortic arch/carotid anatomy will limit the incidence of neurologic events during CAS.

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